Vitamin E Deficiency in Dogs

A VITAMIN E defiency in puppies of litters from dogs maintained on a diet of commercially evaporated milk, characterized by a progressive muscle paralysis, has been reported.

Symptoms of muscular weakness and paralysis in the fore and hind legs were observed about the twentieth day after birth. A general lack of muscle tone and hypersensitivity to pain seemed quite characteristic, together with a marked denudation of the head and limbs. X-ray photographs revealed no faulty bone development.

At the same time the dams began to exhibit signs of a deficiency, with a marked loss of weight, and the development of sores on the teats and other areas subjected to abrasion.

By feeding vitamin E (synthetic a - tocopherol) to the pups and the dams, symptoms were alleviated and recovery took place, if the therapy was initiated before the symptoms became too far advanced.

This deficiency has been reported in experimental studies on rats, guinea pigs and rabbits, but this is the first instance on record of the deficiency in dogs.

*H D. Anderson, E. A. Elevhjem and J. E. Gonce, Jr.
Proc. Soc. Exp. Biol. and Med., Vol. 42. No. 3, 1939, page 750.

CASE REPORTS

Parasitic Aneurysm of the Coeliac Axis

By S. MURRAY*.

TWENTY or thirty years ago, it was not uncommon in this district, at least, to find on the post-mortem examination of a horse that died of colic or enteritis a parasitic aneurysm of the anterior mesenteric artery, due to the *Strongylus armatus*, causing clots and obstructing the branching arteries. Veterinarians who observe their cases closely and who have been fortunate enough to hold post-mortems on a few of these cases, following constant attention in the treatment of them prior to their death, can, with reasonable accuracy, diagnose a typical one.

Unfortunately, at the present time, the practitioner does not have the opportunity to hold equine post-mortems as frequently as in the past as the owner does not wish to go to the trouble of burying the carcass for he can dispose of it to fertilizer plants who, in turn, prefer getting the carcasses intact. This results in neglecting post-mortem examinations. I am sure, however, that no practitioner whose uppermost thoughts are for the interest of the profession will begrudge the time spent on a post-mortem and will, in doubtful and extraordinary cases, insist on being allowed to proceed with an autopsy.

Three weeks previous to the preparation of this report, I was called to a sick two-year-old stallion colt of Clyde breeding. He was recently broken to harness. The first unusual thing the owner noticed was that he became fatigued easily, although in fair flesh. Secondly, he had a ravenous appetite, but only ate about half of his meal, and then suddenly discontinued his feed without showing any signs of pain. A third peculiarity was that he was unable to rise without assistance.

When examined, I found him down lying on his side and unable to rise even with the assistance of the owner and some neighbours. Temperature normal, considerable dyspnoea, but only when exerted. Pulse full, tense, and very fast. Bowels, regular enough, but the faeces consisted of small, dry pellets. Urine, scant and when catheterized, the bladder was almost empty. The urine obtained in this way was highly coloured and of a greenish hue. The mucous membranes were yellow. Slings were used to get the colt up, but

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